

# Epistaxis

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## Epistaxis

### Introduction:

Epistaxis is defined as bleeding from the nasal cavity. It is actually a Greek word for nose bleed. It is actually a very common problem and hence its incidence is rather difficult to access. Crude estimates of its incidence ranges from 5 – 14%<sup>1</sup>. The incidence of epistaxis also shows significant increase during winter months / hot dry climates with low humidity. This climatic increase in incidence of epistaxis has been attributed to increase in the incidence of upper respiratory tract infections. Forceful blowing of inflamed nasal mucosa provokes epistaxis in these patients<sup>2</sup>. Classically epistaxis is known to manifest bimodal incidence – with peaks in age groups of 2-10 and 60-80. Only a small percentage of this population seek Otolaryngologist intervention. This amounts to about 1% of all patients with nasal bleed<sup>3</sup>.

### History:

Epistaxis has been a centre of all folklores. Some associated epistaxis with “Love” while others believed that it foretold death / some form of severe illness. On the whole spirits were believed to cause epistaxis. Lupton in 1601 suggested that the patients use their own blood from epistaxis to write the words “*consummatum est*” on the forehead in order to avoid further episodes. These words were uttered by Jesus Christ as he was dying on the cross. The exact meaning of these words mean “*Its finished*”. Moncrief in 1716 fried the patient's own epistaxis blood and applied the same as snuff as treatment. Hippocrates one of the earliest physicians appreciated that pressure on alae nasi in patients with epistaxis managed to stop the nasal bleed. Ali Ibn Rabban Al-Tabiri<sup>4</sup> a Persian Hakim (850 A.D.) wrote in his classic treatise that epistaxis was due to swelling of vein (Retro columellar) and its eventual rupture. Giovanni Battista Morgagni an Italian Anatomist observed turgid blood vessels located about a finger's breadth from the anterior nasal cavity. He attributed epistaxis to bleeding from this area.

Carl Michel, James Lawrence Little and Kiesselbach identified venous plexus over the anterior part of cartilagenous septum as a source of epistaxis. In 1868 Pilz performed the first documented common carotid artery ligation as treatment of epistaxis. Alfred Seiffert in 1928 introduced the concept of ligation of internal maxillary artery via trans antral approach as a treatment modality for epistaxis. It was Henry Goodyear in 1937 who first ligated anterior ethmoidal artery to treat epistaxis.

### Vascularity of nasal mucosa:

Nasal mucosa is highly vascular. The submucosal blood vessels of nasal cavity receive blood supply from both interanal and external carotid systems. The general rule of thumb is that the area of nasal cavity below the level of middle turbinate is supplied by external carotid branches while the area above the level of middle turbinate<sup>5</sup> is supplied by internal carotid artery. Anastomosis between these two systems are known to occur within the nasal cavity. It should be borne in mind

that the pressure levels at the internal carotid artery is higher than that of external carotid artery.

External carotid system: Blood from the external carotid system reaches the nasal cavity via the facial and the internal maxillary arteries which are branches of the external carotid artery. The artery of epistaxis is the sphenopalatine branch of internal maxillary artery. This is called so because this vessel supplies the major portion of the nasal cavity. It enters the nasal cavity at the posterior end of the middle turbinate to supply the lateral nasal wall, it also gives off a septal branch which supplies the nasal septum.

Facial artery: the superior labial branch of the facial artery is one of its terminal branches. It supplies the anterior nasal floor and anterior portion of the nasal septum through its septal branch.

Internal maxillary artery: after entering into the pterygopalatine fossa this vessel gives rise to 6 branches. These branches are posterior superior alveolar artery, descending palatine artery, infra orbital artery, sphenopalatine artery, pterygoid artery, and pharyngeal artery. The descending palatine artery enters the nasal cavity through the greater palatine canal to supply the lateral wall of the nose, it also contributes blood supply to the nasal septum through its septal branch.

Internal carotid system: the internal carotid artery supplies the nasal cavity via its ophthalmic artery. It enters the orbit via the superior orbital fissure and divides into many branches. The posterior ethmoidal artery one of the branches of ophthalmic artery exits the orbit via the posterior ethmoidal foramen located 2-9 mm anterior to the optic canal. The anterior ethmoidal artery which is larger leaves the orbit through the anterior ethmoidal foramen. Both these vessels cross the roof of the ethmoid and descends into the nasal cavity through the cribriform plate. It is here that these vessels divide into lateral and septal branches to supply the nose.

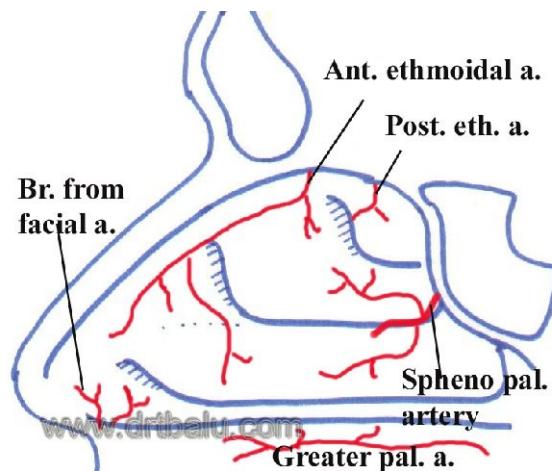


Figure showing blood supply of lateral nasal wall

Little's area: This area is located in the anterior part of the cartilagenous portion of the nasal septum. Here there is extensive submucous anastomosis of blood vessels both from the external and the internal carotid systems. Bleeding commonly occurs from this area since it is highly vascular and is

also exposed to the exterior. Anastomosis occur between the septal branches of sphenopalatine artery, greater palatine artery, superior labial artery and the anterior ethmoidal artery. This plexus is also known as Keisselbach's plexus. Bleeding from this area is common because mucosal drying occurs commonly here and this area is easily accessible to nose picking. Among the vessels taking part in the anastomosis the anterior ethmoidal artery is from the internal carotid system while the other vessels are from the external carotid system. Bleeding from this area is clearly seen and easily accessible and flows through the anterior nasal cavity hence it is known as anterior bleed.

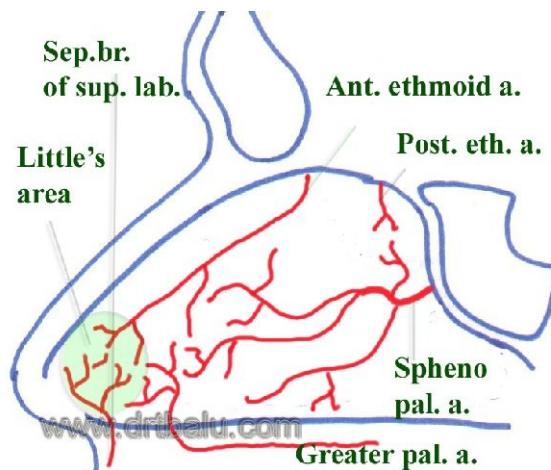


Illustration showing Little's area

Two areas have been implicated in epistaxis. Little's area has been implicated in anterior epistaxis and Woodruff's plexus in posterior epistaxis. Anterior epistaxis is common in children while posterior epistaxis is common in adults.

Woodruff's plexus: is responsible for posterior bleeds. This area is located over the posterior end of the middle turbinate. The anastomosis here is made up of branches from the internal maxillary artery namely its sphenopalatine and ascending pharyngeal branches. The maxillary sinus ostium forms the dividing line between the anterior and posterior nasal bleeds. Posterior nasal bleeds are difficult to treat because bleeding area is not easily accessible. Bleeding from Woodruff's plexus commonly occur in patients with extremely high blood pressure. Infact this plexus acts as a safety valve in reducing the blood pressure in these patients, lest they will bleed intracranially causing more problems. In patients with posterior bleeds it is difficult to access the amount of blood loss because most of the blood is swallowed by the patient.

Woodruff in 1949 reported a group of large blood vessels in the lateral wall of inferior meatus posteriorly. He was able to visualize these blood vessels using a rigid nasopharyngoscope. He coined the term "Naso nasopharyngeal plexus" to describe these vessels. He suspected the association between the presence of these dilated blood vessels and posterior epistaxis. He was not sure whether these vessels are veins or arteries.

Microdissection studies revealed a superficial collection of fragile fairly large calibre blood vessels lying just beneath the surface mucosa<sup>6</sup>. There was very little intervening connective tissue. Histological studies revealed that the epithelium overlying the posterior inferior meatus was typical respiratory epithelium. The blood vessels in this area were sinus like with very little muscle or fibrous tissue within their walls. The average blood vessel diameter in this area is 1-2mm.

Shaheen described Woodruff's plexus as an arterial plexus formed by anastomosis between pharyngeal, posterior nasal, sphenopalatine and posterior septal arteries. Microdissection and histological studies have proved Woodruff's plexus to be venous in origin.

Bleeding from the blood vessels of Woodruff's plexus could result in a slow but prolonged ooze. Since these blood vessels have no muscle walls, hemostasis is poor. Post nasal packing will have to be resorted to in rare cases to stop bleeding.

**Etiology:** The etiology of epistaxis is not just simple or straight forward. It is commonly multifactorial, needing careful history taking and physical examination skill to identify the cause. For purposes of clear understanding the etiology of epistaxis can be classified under two broad heads, i.e. local and systemic causes<sup>7</sup>.

**Local factors causing epistaxis:** include vascular anomalies, infections and inflammatory states of the nasal cavity, trauma, iatrogenic injuries, neoplasms and foreign bodies. Among these causes the commonest local factors involved in epistaxis is infection and inflammation. Infections and inflammation of the nasal mucous membrane may damage the mucosa leading on to bleeding from the underlying exposed plexus of blood vessels. Chronic granulomatous lesions like rhinosporidiosis can cause extensive epistaxis.

Aneurysms involving the internal carotid artery may occur following head injury, injury sustained during surgical procedures. These extradural aneurysms and aneurysms involving the cavernous sinus may extend into the sphenoid sinus wait for the opportune moment to rupture. It can cause sudden fatal epistaxis, or blindness. Urgent embolisation is the preferred mode of management of this condition.

Trauma is one of the common local causes of epistaxis. It is commonly caused by the act of nose picking in the Little's area of the nose. This is commonly seen in young children. Acute facial trauma may also lead to epistaxis. Patients undergoing nasal surgeries may have temporary episodes of epistaxis.

**Irritation of the nasal mucous membrane:** any disruption of normal nasal physiology can cause intense drying and irritation to the nasal mucosa causing epistaxis. These episodes are common during extremes of temperature when the nasal mucosa is stressed to perform its airconditioning role of the inspired air. In these conditions there is extensive drying of nasal mucosa causes oedema of the nasal mucous membrane. This oedema is caused due to venous stasis. Ultimately the mucosa breaches exposing the underlying plexus of blood vessels causing epistaxis.

**Anatomical abnormalities:** Common anatomical abnormality causing epistaxis is gross septal deviation. Gross deviations of nasal septum causes disruption to the normal nasal airflow. This disruption leads to dessication / drying of the local mucosa. The dry mucosa cracks and bleeds.

**Septal perforations:** Chronic non healing septal perforations can cause bleeding from the granulation tissue around the perforation.

**Neoplasms:** involving the nose and paranasal sinuses can cause epistaxis. Neoplasms include benign vascular tumors like hemangioma, juvenile nasopharyngeal angiofibroma, and malignant

neoplasms like squamous cell carcinoma. If epistaxis occurs along with secretory otitis media then nasopharyngeal carcinoma should be the prime suspect.

Systemic causes for epistaxis:

Hypertension is one of the common systemic causes of epistaxis. Accumulation of atherosclerotic plaques in the blood vessels of these patients replaces the muscular wall. This replacement of muscular wall reduces the ability of the blood vessels to constrict facilitating epistaxis. This is one of the common causes of posterior nasal bleeds. It commonly arises from the Woodruff's plexus found close to the posterior end of the middle turbinate. Beran et al concluded that hypertension had no significant impact as etiological factor for epistaxis <sup>7</sup>.

Hereditary hemorrhagic telangiectasia also known as Osler – Rendu – Weber disease is another systemic disorder known to affect the blood vessels of the nose. This is an autosomal dominant non sex linked disorder. This disease causes loss of contractile elements within the blood vessels causing dilated venules, capillaries and small arteriovenous malformations known as telangiectasia. These changes can occur in the skin, mucosal lining the whole of the respiratory passage and urogenital passage. Bleeding from these telangiectasia is difficult to control. Bleeding invariably starts when the patient reaches puberty. Common cause of mortality in these patients is gastrointestinal bleed.

This condition is more common in women 5:1. Serious epistaxis is known to occur in nearly 80% of these patients by the age of 30 <sup>8</sup>. Depending on the amount of blood loss decision on transfusion is made. Nasal packing and cauterization is advocated for mild to moderate bleeding in these patients. Treatment of this condition is rather palliative since the underlying disease is not curable <sup>9</sup>. Laser coagulation of bleeders have been tried out with reasonable degree of success in these patients. Topical oestrogen therapy following laser cauterization of bleeders help in squamous metaplasia of nasal epithelium thereby reducing the incidence of bleeding <sup>10</sup>. In very refractory cases nasal obliteration too has been attempted (Young's procedure).

Systemic diseases like syphilis, tuberculosis & Wegener's granulomatosis cause epistaxis because of their propensity to cause ulceration of the nasal mucous membrane. Viral infections like dengue and haemorrhagic fever cause epistaxis due to reduced platelet count.

Blood dyscrasias can also cause epistaxis. A low platelet count is one common cause of nasal bleed in this category. In thrombocytopenia the platelet count is less than 1 lakh. Epistaxis can start when the platelet count reduces to 50,000. Platelet deficiency can be caused by ingestion of drugs like aspirin, indomethacin etc. Hypersplenism can cause thrombocytopenia in idiopathic thrombocytopenic purpura. These patients need to be transfused fresh blood in adequate quantities. Only when the platelet count increases will the nasal bleed stop.

**Incidence:** The incidence of epistaxis is known to be slightly higher in males. It also has a bimodal distribution affecting young children and old people.

**Evaluation:** While evaluating a patient with epistaxis it is absolutely necessary to assess the quantum of blood loss. The blood pressure and pulse rate of these patients must be constantly monitored. These patients will have tachycardia. Infusion of fluid must be started immediately.

Initially ringer lactate solution will suffice. If the patient has suffered blood loss of more than 30% of their blood volume (about 1.5 liters) then blood transfusion becomes a must. Further examination should be started only after the patient's general condition stabilises.

**History:** Careful history taking is a must. History taking should cover the following points:

1. History regarding the frequency, severity and side of the nasal bleed.
2. Aggravating and relieving factors must be carefully sought.
3. History of drug intaken must be sought.
4. History of systemic disorders like hypertension and diabetes mellitus must be sought.

**Physical examination:**

The nasal pack if any must be removed. Anterior nasal examination should be done, first attempted without the use of nasal decongestants. If visualisation is difficult due to oedema of the nasal mucosa then nasal decongestants can be used to shrink the nasal mucosa. The solution used for anesthetising the decongesting the nose is a mixture of 4% xylocaine and xylometazoline.

Nasal endoscopy can be performed under local anaesthesia to localise posterior bleeds.

**Investigations:**

If bleeding is minimal no investigation is necessary.

If bleeding is more than a complete blood work up to rule out blood dyscrasias is a must. It includes bleeding time, clotting time, platelet count and partial thromboplastin time.

Imaging studies like CT scan of the para nasal sinuses must be done to rule out local nasal conditions of epistaxis. Imaging must be done only after 24 hours of removing the nasal packing. Scans done with the nasal pack or immediately after removing the nasal pack may not be informative.

In difficult and intractable cases angiography can be done and the internal maxillary artery can be embolised in the same sitting. This procedure should be reserved only for cases of intractable nasal bleeding.

**Classification of etiological factors of epistaxis:**

For better understanding etiological factors of epistaxis has been classified under two heads:

1. Local causes
2. Systemic causes



## Local Causes

Trauma	Pressure Changes	Nasal Dryness	Septal Perforation	Chemical	Tumours
Inflammation					

Trauma:

This is the most common cause of epistaxis. This category includes:

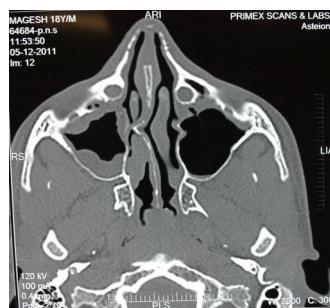
Fractures

Self induced digital trauma / foreign body

Iatrogenic – surgical procedures involving nose and sinus and skull base

Barometric changes – Extremes of atmospheric pressure changes can cause epistaxis.

Nasal dryness: This is caused either by dry air or a combination of dry air with septal deviations.



CT scan showing acute deviation of nasal septum



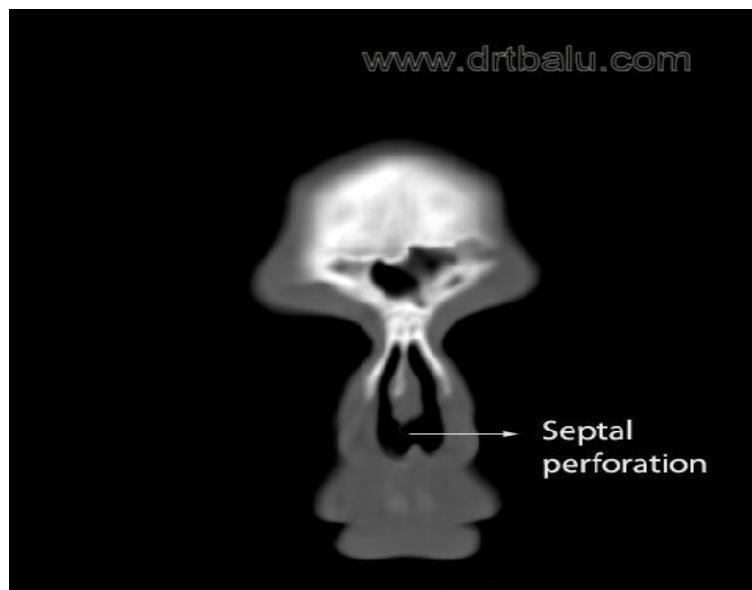
Fracture nasal bone being repaired

### Septal perforation:

This is one of the local cause of epistaxis. Perforated nasal septum causes excessive drying of edges of the perforation causing bleeding from the nose.



Image showing septal perforation



Coronal CT scan of nose showing septal perforation

Exposure to chemicals:

Cocaine abuse

Steroid spray

Decongestants (Rhinitis Medicamentosa)

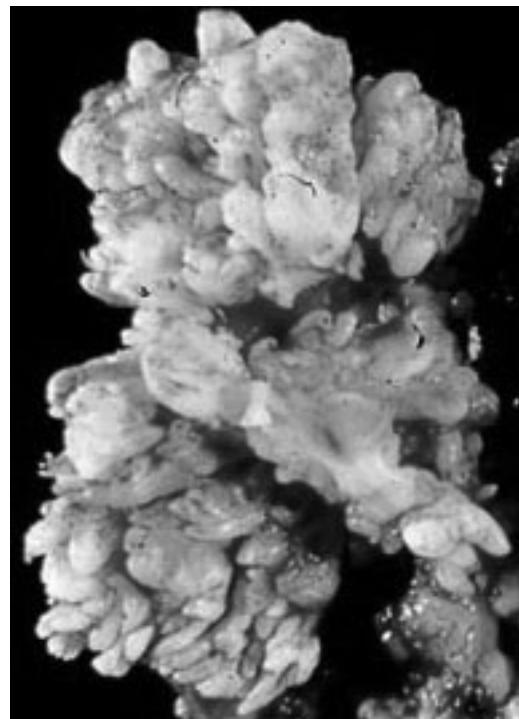
Ammonia

Gasoline fumes, Chromium salts, Sulfuric acid

Tumors:

Benign – Inverted papilloma, JNA, Septal angioma

Malignant – Squamous cell carcinoma, esthesioneuroblastoma



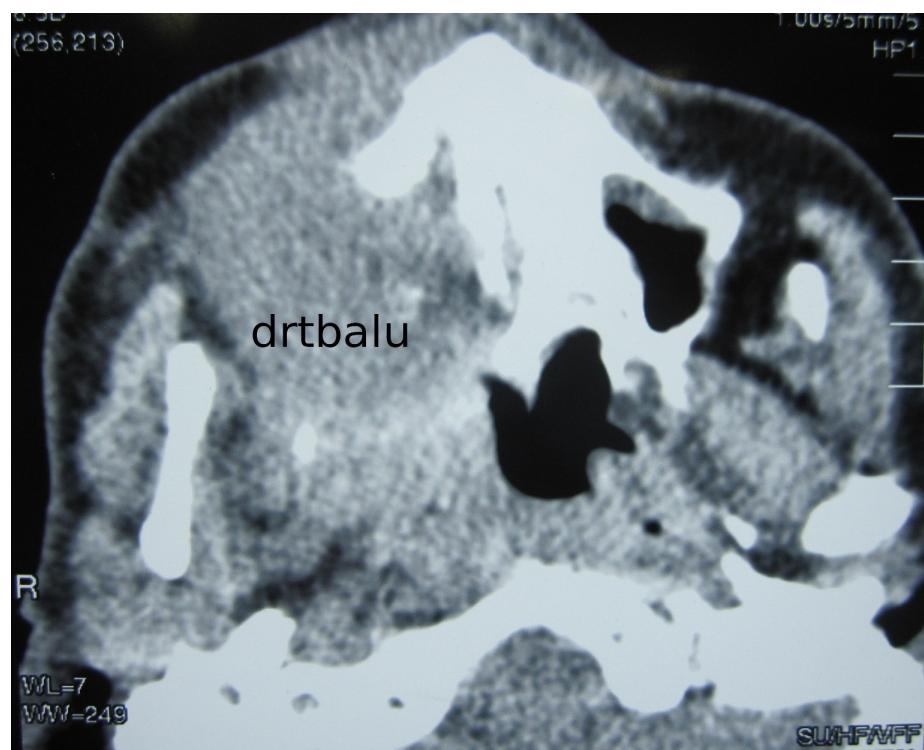
Inverted Papilloma



Mass right nasal cavity



Image showing JNA mass



CT scan showing malignant growth right maxilla with extensive destruction of anterolateral wall of maxilla and involvement of pterygopalatine fossa

Inflammation:

Presence of mucosal inflammation will cause epistaxis in these patients.

Rhinitis

Sinusitis

Atrophic rhinitis<sup>14</sup> which is characterised by crusting of nasal mucosa, foul smelling nasal discharge, anosmia also causes epistaxis.

Rhinosporidiosis<sup>15</sup> caused by infection with Rhinosporidium seeberi presents with epistaxis.

Rhinolith



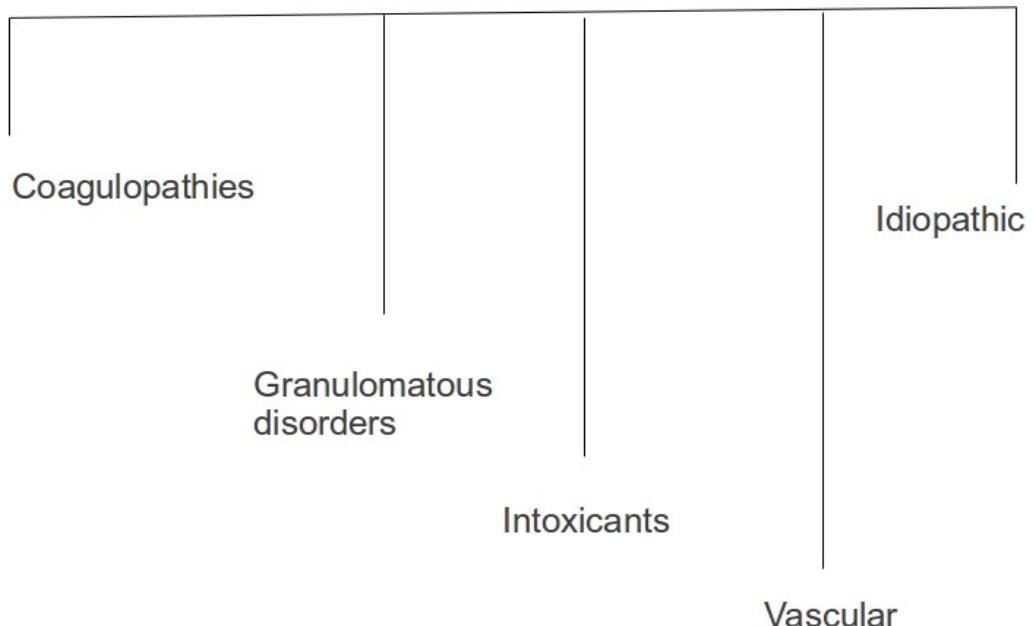
Endoscopic view of a patient with atrophic rhinitis



Rhinosporidial mass occupying the right nasal cavity

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## Systemic Causes



Systemic causes:

Coagulopathies:

Anticoagulant / antiplatelet drug use

Haemophilia

Platelet defects

Pregnancy

Hepatic insufficiency

Alcohol ingestion

Scurvy

Haemorrhagic fevers

Granulomatous disorders – Wegener's disease, Midline granuloma, Syphilis, Tuberculosis, Rhinoscleroma, SLE, Periarteritis nodosa



Image showing granulomatous lesion of nose

Intoxicants:

Cobalt

Phosphorous

Arsenic

Lead

Vascular causes:

Hypertension

Atherosclerosis

Hereditary Haemorrhagic Telangiectasia



Coronal CT showing rhinolith involving right nasal cavity

Management:

General assessment:

1. Assessment of airway
2. Vitals to be checked
3. Blood grouping and cross matching

Pinching the nose:

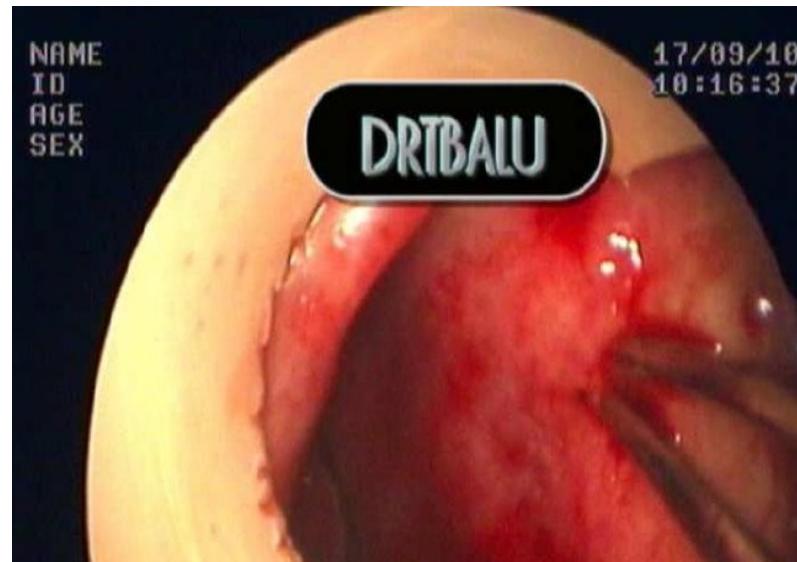
This is a useful first aid measure in patients with anterior episataxis. The patient is asked to pinch the nose while leaning forwards. Swimmer's nose clip<sup>12</sup> can also be used for this purpose. This provides constant localised pressure over the bleeding point and obviates the need to keep the nose pinched manually.



Image showing swimmer's nose clip

Management of anterior nasal bleed:

If the bleeding arises from Little's area / retrocolumellar vein area then cauterization may be resorted to. Bipolar cautery is ideal. In outpatient setup chemical cautery (Chromic acid, acetic acid) can also be tried out. Silver nitrate even though commonly used is not effective during active bleeds. Studies show that 30 seconds of exposure to chemical agent causes 1 mm penetration of tissue<sup>16</sup>. Cauterization should be precise. Random cauterization should be avoided at all costs as this could lead to troublesome septal perforation. After effective cauterization of the bleeding point patient is advised to use saline nasal spray to prevent excessive drying of nasal mucosa.



Bipolar cautery in action

#### Use of nasal cream:

In children the most common cause of bleeding is from enlarged retrocolumellar vein. Constant nose picking could cause bleeding from this area. This area lies slightly anterior to the Little's area. Application of antiseptic cream <sup>13</sup> in this area would reduce the incidence of nasal picking in these children.

#### Topical Haemostats:

Many topical haemostatic agents are available at present. These agents exert their effect by:

1. Improving primary haemostasis
2. Stimulating fibrin formation
3. Inhibiting fibrinolysis
4. Provides template for maintenance of endogenous coagulation

## Types of Topical Haemostats:

1. Collagen based
2. Gelatin based
3. Cellulose based
4. Albumin derived
5. Inorganic haemostats
6. Fibrin based
7. Polymeric haemostats

### Collagen based:

These haemostats were first introduced in 1970. These substances possess microfibrillar structure comprising of collagen molecules with non covalently bound hydrochloric acid. The molecular structure and the large surface area it provides are important for achieving haemostasis <sup>24</sup>. Contact with the bleeding area attracts platelets which gets entangled within the microfibrillar structure and degranulates there by promoting coagulation.

### Gelatin based:

The mechanism of action of gelatin based topical haemostasis is not clearly understood. It promotes coagulation because of its surface effects. This may also be used alone or in combination with procoagulants. Floseal is a characteristic example. It contains gelatin based topical haemostatic and a procoagulant.

### Cellulose based:

These have been in use for more than decades. Mechanism of action include:

1. Absorption of blood
2. Surface interactions with proteins and platelets
3. Activation of both intrinsic and extrinsic pathways <sup>25</sup>

### Albumin derived:

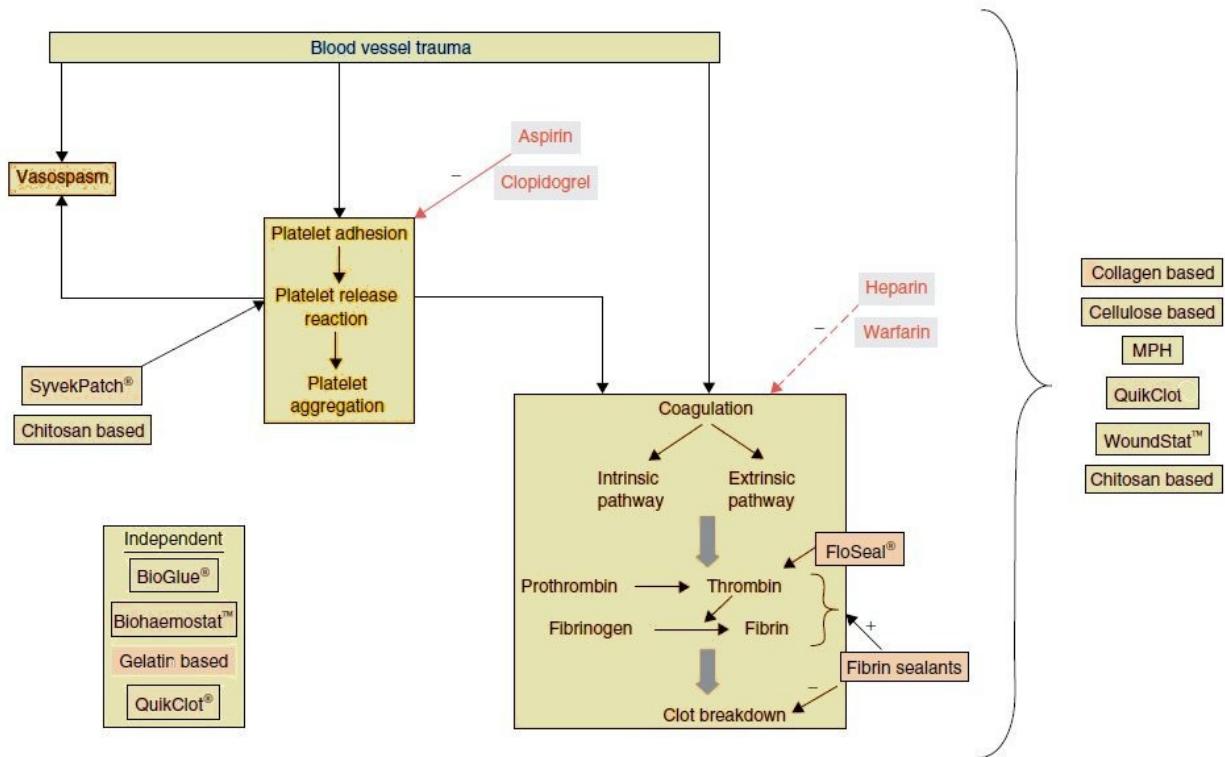
Classic example of drug belonging to this category is gelatin–resorcinol–formaldehyde . It has been abandoned now due to toxic reactions caused by formaldehyde. Another haemostat of this category which is commonly used is Bioglue. In bioglue formaldehyde is replaced by less toxic gluteraldehyde. Advantage of this category of topical haemostats include its ability to cause coagulation outside both extrinsic and intrinsic pathways.

### Ionic based:

This is actually a recent addition to the haemostat armamentorium. Most commonly used is Quickclot. This is based on zeolite. The mode of action is absorption of water from the bleeding site causing an increase in the concentration of platelets and coagulation factors.

### Fibrin based:

Fibrin based tissue adhesives has been in use since 1970's. They have both hameostatic and adhesive properties and can be used to deliver antibiotics to the wound site. They have been reported to reduce adhesion formation and enhance wound healing.



## Diagram showing various sites of action of Topical haemostats

## Use of Gelfoam / Surgicel:

Absorbable haemostatic material like Gelfoam / surgicel can be used to pack the nasal cavity. This is very useful in managing patients with coagulopathy. Since this pack need not be removed mucosal trauma during removal can be avoided in these patients.

Gelfoam is actually a sterile haemostatic sponge prepared from purified Porcine skin gelatine. This is actually water insoluble. It has the capacity to absorb 45 times its weight of blood<sup>17</sup>. Hence its absorptive capacity is directly proportional to its size<sup>18</sup>. The mechanism of haemostatic action of gelfoam is supportive and mechanical in nature. When applied to bleeding surfaces these substances arrest bleeding by forming artificial clots thereby providing the mechanical matrix that facilitates clotting mechanism<sup>19</sup>. Clotting effects of gelfoam is due to release of thromboplastin by Platlets that come into contact with gelfoam<sup>20</sup>. Gelfoam pack when applied to nasal cavity completely liquifies within 2-5 days.



Surgicel

#### Conventional nasal packing:

This is indicated in patients where cauterization is not possible / fails. Conventional nasal packing are of two types: Anterior nasal packing and post nasal packing.

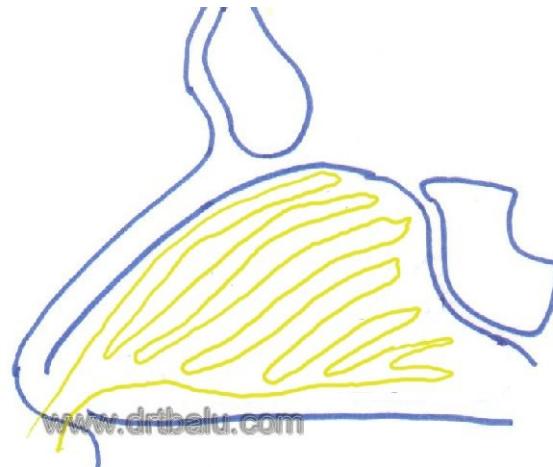
#### Materials used for nasal packing:

1. Roller gauze impregnated with antibiotic and lubricant like liquid paraffin
2. Merocel
3. Tampons
4. Foley's catheter for post nasal packing

If gauze is used for packing nasal cavity then it should be removed within 48 hours. If left in place for more days there is always an associated risk of nasal infection and toxic shock syndrome. If post nasal packing is resorted to then the patient should be admitted for close monitoring of oxygen saturation and airway observation.

#### Complications:

1. Headache
2. Obstruction to sinus drainage
3. Epiphora



Anterior nasal packing

Anterior nasal packing using roller gauze impregnated with liquid paraffin is sufficient to manage a majority of anterior nasal bleeds. The liquid paraffin acts as a lubricant, and as a moistening agent. The tamponading effect of a nasal pack is sufficient to stop nasal bleeding. This type of roller gauzes can be kept inside the nasal cavity only up to 48 hours after which it has to be removed and changed. The newer packs like the BIPP (Bismuth Iodine paraffin paste) packs can be left safely in place for more than a week.



Merocel

To manage post nasal bleed a post nasal pack is a must. Post nasal packing can be done in 2 ways:

Post nasal packing (conventional): A gauze roll about the size of the patient's naso pharynx is used here. Three silk threads must be tied to the gauze roll. One at each end and the other one at the middle. The patient should be in a recumbent position. After anesthetising the nasal cavity with 4% xylocaine the mouth is held open. Two nasal catheters are passed through the nasal cavities till they reach just below the soft palate. These lower ends of the catheters are grasped with forceps and pulled out through the mouth. The silk tied to the ends of the gauze is tied to the nasal catheters. The post nasal pack is introduced through the mouth and gradually pushed into the nasopharynx, at the same time the nasal catheters on both sides of the nose must be pulled out. When the pack snugly sits inside the nasopharynx, the two silk threads tied to its end would have reached the anterior nares along with the free end of the nasal suction catheter.

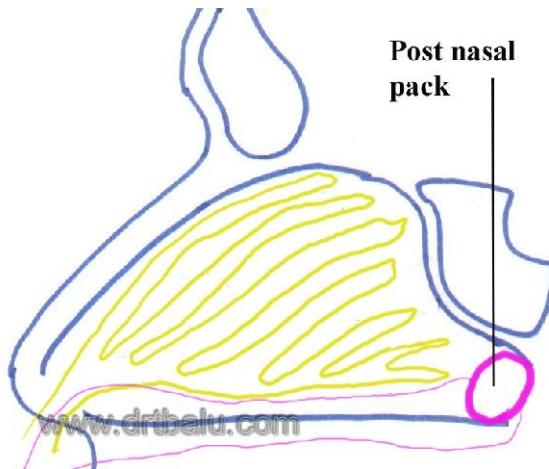


Figure showing post nasal pack along with anterior nasal pack

The two silk threads tied to the suction catheters are untied. The catheters are removed from the nose. The silk thread is used to secure the pack in place by tying both the ends to the columella of the nose. The silk tied to the middle portion of the gauze pack is delivered out through the oral cavity and taped to the angle of the cheek. This middle portion silk will help in removal of the nasal pack. In addition to the postnasal pack anterior nasal packing must also be done in these patients.

Postnasal pack using balloon catheters: Specially designed balloon catheters are available. This can be used to perform the post nasal pack. Foleys catheter can be used to pack the post nasal space. Foley's catheter is introduced through the nose and slid up to the nasopharynx. The bulb of the catheter is inflated using air through the side portal of the catheter. Air is used to inflate the bulb because even if the bulb ruptures accidentally there is absolutely no danger of aspiration into the

lungs. After the foleys catheter is inflated the free end is knotted and anchored at the level of the anterior nares.

Newer packing materials: Newer packing materials made of silicone are available. The advantages of these material are that they are not irritating, patient can breath through the nose with the pack on through the vent provided, these packs can be retained inside the nasal cavity for more than 2 weeks. They can be removed and repositioned if necessary. The only disadvantage is that they are expensive.



Figure showing Foley's catheter



Baloon tamponades

### Surgical management:

Endoscopic cauterisation can be tried if the bleeders are localised and accessible. If not accessible, ligation of the internal maxillary artery can be done through caldwelluc approach. Sphenopalatine artery clipping can be done endoscopically. It is accessible close to the posterior end of the middle turbinate. In rare cases external carotid artery ligation at the neck can be resorted to. External carotid artery is differentiated from the internal carotid in the neck by the fact that internal carotid artery does not give rise to branches in the neck, while the external carotid artery does so.

Ethmoidal artery ligation: If epistaxis occur high in the nasal vault, anterior and posterior ethmoidal arteries may be ligated using ligoclips. These arteries can be accessed using an external ethmoidectomy incision. The anterior ethmoidal artery is usually found 22mm from the anterior lacrimal crest. If ligation of the anterior ethmoidal artery does not stop bleeding then posterior ethmoidal artery should also be ligated. The posterior ethmoidal artery can be found 12mm posterior to the anterior ethmoidal vessel.

### TESPAL:

Tespal: (Trans nasal endoscopic sphenopalatine artery ligation)

History: This procedure was first reported by Budrovich and Saetti in 1992.

This procedure can safely be performed under GA. / L.A.

#### Indication:

Epistaxis not responding to conventional conservative management.

Posterior epistaxis

#### Procedure:

The nose should first be adequately decongested topically using 4% xylocaine mixed with 1 in 50,000 units adrenaline.

A 4mm 0 degree nasal endoscope is introduced into the nasal cavity. The posterior portion of the middle turbinate is visualized. 2% xylocaine with 1 in 1lakh units adrenaline is injected in to this area to further reduce bleeding.

Incision: An incision ranging between 10 - 20 mm is made vertically about 5 mm anterior to the attachment of the middle turbinate. The mucosal flap is gently retracted posteriorly till the crista ethmoidalis is visualized. The crista ethmoidalis is a reliable land mark for the sphenopalatine artery. The artery enters the nose just posterior to the crista. The crista can in fact be removed using a Kerrison's punch for better visualization of the artery.

The sphenopalatine artery is clipped using liga clip or cauterized as it enters the nasal cavity. This is done as close to the lateral nasal wall as possible, this would ensure that the posterior branches may also be reliably included.

Following successful ligation / cauterization, the area is explored posteriorly for 2 - 3 mm to ensure that no more vessels remain uncauterized.

Nasal packing is not needed.

Complications of TESPAL:

1. Palatal numbness
2. Sinusitis
3. Decreased lacrimation
4. Septal perforation
5. Inferior turbinate necrosis

This procedure in combination with transnasal anterior ethmoidal artery ligation ensures that epistaxis is controlled reliably.

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